Cardiac Response to Exercise in Normal Ageing: What Can We Learn from Masters Athletes?

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Abstract

Ageing is associated with a progressive decline in cardiac and vascular health, resulting in an increased risk of cardiovascular disease (CVD). Lifestyle factors such as exercise have emerged as a primary therapeutic target in the prevention of CVD, yet older individuals are frequently reported as being the least active, with 1 in four adults failing to meet the physical activity guidelines. In contrast, well trained older individuals (Masters athletes) have superior functional capacity than their sedentary peers and are often comparable with young non-athletes. Therefore, the decline commonly observed in untrained older individuals overestimates the decline due to age *per se* and masters athletes represent a viable model by which to determine the degree to which functional capacity may be maintained, and the extent of the unavoidable 'true' reduction in functional capacity due to ageing. This review examines evidence from observational studies which have compared well trained older athletes, with age matched, sedentary, but otherwise healthy controls, and the consequences on cardiac structure and function are discussed.

Keywords: systolic function, diastolic function, cardiac remodelling, healthy ageing,

1 Introduction

Improvements in long-term survival from previously fatal conditions have increased the number of older, and elderly individuals worldwide. Indeed, the World Health Organisation (WHO) estimates that the number of individuals aged over 60 years of age has doubled since 1980, and will triple by 2050 (1). Consequently, ensuring that increases in lifespan occur in tandem with increases in health span is emerging as a critical public health challenge of our generation. In addition, despite significant improvements in the treatment of cardiovascular disease (CVD), it remains the main cause of mortality worldwide (2) and accounts for almost one third of deaths globally (3). Moreover, for those who survive with CVD, there are substantial costs both financially (4) and in reduced quality of life and reduced functional capacity (5). Furthermore, while advances in healthcare and survival are welcome, the increase in the number of people coping with the daily challenges of cardiovascular morbidity are substantial. In the UK alone there are an estimated 7 million people coping with ongoing CVD, requiring more than £9 billion in healthcare costs (6).

Modifiable lifestyle factors such as exercise and physical activity (PA) have emerged as a primary therapeutic target in the prevention of CVD, with extensive epidemiological, pre-clinical, and human interventional studies to support its efficacy (7). Multiple lines of evidence indicate that those individuals who are most active enjoy superior cardiac function, as well as lower levels of systemic inflammation and oxidative stress (8–11). Correspondingly, this has resulted in a wealth of health promotion recommendations promoting PA for both children and adults (12,13). Despite these such recommendations, older individuals are frequently reported as being the least active, with 1 in four adults failing to meet the weekly PA guidelines for health worldwide, of at least 150 minutes moderate or

75 minutes of vigorous intensity exercise (14), rising to 85 to 90% of older adults in many developed countries (15).

In contrast, investigations of ageing athletes frequently report that relative to their sedentary counterparts, they exhibit high levels of cardiovascular reserve (i.e stroke volume (SV) and maximal cardiac output; (16)) while simultaneously presenting with minimal risk factors for CVD (11). In studies of cardiovascular function, endurance trained masters athletes have superior functional capacity, cardiovascular reserve, than their sedentary peers, and which are comparable with much younger non-athletes (17,18). In this respect, the 'masters athlete' may be viewed as a unique non-pharmacological model which may allow researchers to disentangle the inexorable from the preventable effects of ageing on cardiac and vascular health. A masters or veteran athlete has been defined as an individual older than 45 or 50 years of age competing regularly in endurance events (19,20). Since a meta-analysis has been performed on cardiac structural and functional adaptation in in younger athletes up to 45 years of age (21), the present review aims to summarise the available literature regarding the effect of exercise on cardiac health in normal ageing, and with specific reference to comparisons between older sedentary individuals and masters athletes $\geq$ 45 years of age.

2 Exercise, Ageing and Cardiac Function

2.1 LV Diastolic Function

2.1.1 The Impact of Healthy Ageing on Diastolic Function

Diastolic function may be divided into 2 components, compliance and relaxation (22). Myocardial relaxation concerns myocyte calcium handling, whereas ventricular compliance

is determined by the interaction between compliant cardiac muscle and less compliant (stiff) connective tissue and extracellular matrix (22). The inevitability of chronological sedentary yet, healthy ageing seemingly leads to a gradual decline in LV compliance until approximately 64 years of age, at which point LV stiffening may be deemed complete (23). Similarly, with progressive age, early diastolic function determined by Doppler indices of LV diastolic function show reduced early (E) inflow velocity, ratio of early-to-late inflow velocity (E/A) (24), early diastolic tissue velocity (e'), and gradual increases in the isovolumic relaxation time (IVRT) and time constant of isovolumic pressure decay (Tau) (25). Collectively, these functional changes highlight a worsening of LV diastolic function inherent to the ageing process.

2.1.2 Healthy Ageing and Diastolic Function in Relation to Exercise

Chronic endurance exercise consisting of multiple years of continued training preserves LV compliance (26), which may be reflected in a 'dose' dependant manner (27). In healthy seniors aged >64 years, Bhella *et al.* (27) found an exercise dose of at least 4 to 5 sessions per week, categorised as 'committed exercisers', was sufficient to prevent the age-associated decreases in LV stiffness and distensibility. Nonetheless, 1 year of aerobic exercise training in previously sedentary, older (71 ± 3 years) individuals did not alter LV compliance or stiffness and therefore, it is possible that exercise initiation prior to reaching 'older' age is necessary to reverse the detrimental impact of ageing (28). In support, although mitral inflow and tissue velocity indices were not different between sedentary older men (59 ± 3 years) and exercisers who either began exercising prior to 30 years of age or after 40 years of age, LV end-systolic elastance (E_{LV}) was lower in both trained groups compared with their untrained counterparts, suggesting a less stiff ventricle in the exercise groups (29).

Long term exercise does not prevent the gradual decline in resting global diastolic function associated with ageing, as measured by conventional Doppler mitral inflow or tissue velocities (30–38). Nevertheless, when compared with controls of the same age, older (>45 years) endurance trained athletes have shown improved diastolic function with greater E (19,37–39), e' (19,36), lower late mitral inflow velocity (A) (34–36,40–44), lower late mitral annular tissue velocity (a') (35,45) and collectively, greater e'/a' (19,35,41) and E/A (see Table 1). Equally, in older recreationally active, leisure time athletes, of which sporting discipline was unknown, E/A was greater in trained than untrained (46,47). Indeed, the heart rate and preload dependence of mitral and tissue velocities are known (48,49); bradycardia lengthens the diastolic period and reduces the atrial contribution to filling (35,50). Therefore, it is possible that superior diastolic function in older athletes may be mediated, in part, by a lower heart rate and/or increased plasma volume. In contrast, a significant body of evidence disputes a beneficial influence of endurance based exercise on global diastolic function, expressed as E/A, between age-matched athletes and controls (see Table 1). Thus, it is unclear at present whether exercise is a useful mitigant of the inevitable age-related decline in global diastolic function, when determined by the profiling of mitral inflow velocity. Differences is the participant characteristics and training habits between cross-sectional investigations may well contribute to the conflicting findings.

Soccer specific training of 2 hours per week for 4 months in previously sedentary seniors (68 years of age) sufficiently increased E/A, which was not observed during the equivalent strength-based intervention (51). Similarly, short term training (12 weeks) elicited an increased (52), or demonstrated a trend toward greater (36) E/A in older adults (>62 years) following high intensity interval training (HIIT); whereas, others found no changes in E/A after 8 weeks HIIT (53). Nonetheless, 5 days of intensified training in seniors (68 years of age) resulted in 37% greater E/A, with the change in E/A significantly related to changes in

maximal oxygen uptake ($r=0.52$, $p<0.05$). Despite some unavoidable decline in diastolic function inherent to progressive ageing and irrespective of the mechanistic underpinning of whether changes reflect altered loading conditions, heart rate or intrinsic functional modifications, these data provide some support for improved diastolic function concomitant with exercise training and/or compared with age-matched sedentary counterparts.

The unidimensional motion of tissue velocities do not provide a full description of the LV movements during diastole (38), in comparison to the assessment of LV rotational mechanics, namely untwisting, which can provide further insight into the intrinsic function of the heart at various stages of the cardiac cycle. Recently, in middle-aged (~54-57 years) male athletes, Maufrais *et al.* (35,54) reported no training effect on E or e' at rest but demonstrated significantly greater percentage of untwist during IVRT. This observation is of particular importance considering the percentage of untwist during early diastole declines with age (55), and may therefore suggest a preservation of early diastolic function into old age in aerobic exercisers. Peak untwisting velocity however, is contrasting between studies, with some observing greater in athletes than controls (54) and others finding comparable between trained and untrained groups (34,35). During exercise, Carrick-Ranson *et al.* (31) found the larger SV in older trained men was not the result of faster LV mitral inflow or tissue velocities, with the authors speculating larger LV dimensions with heightened compliance likely responsible. During submaximal exercise, greater E and peak untwisting velocity (54), in addition to a shorter time-to-peak untwisting velocity have been observed in senior trained men than controls (34). Whereas, Lee *et al.* reported unchanged untwisting velocity from rest to exercise in middle-aged trained men, yet an increase in their age-matched untrained counterparts. Nonetheless, since the peak base-to-apex intraventricular pressure gradient is linearly related to peak untwisting velocity (56), superior untwisting mechanics in older trained populations would likely enhance LV filling during exercise when

IVRT and diastole shorten. Still, the heterogeneity between studies warrants further investigation with consideration of characteristics such as training habits, type, intensity, frequency and the number of years engaged in regular training.

Future reporting of speckle tracking echocardiographic (STE) derived untwist mechanics will provide incremental information complimentary to conventionally derived Doppler velocities. Much work is needed to fully elucidate the influence of long term exercise on diastolic function in relation to healthy ageing.

2.2 LV Morphology

Normal ageing is associated with an increase in the LV wall thickness, possibly related to a loss of cardiomyocytes initiating a cellular compensatory processes increasing in cardiomyocyte size (LV hypertrophy) (57). While several cross-sectional studies have documented increased absolute wall thickness in athletes compared with untrained controls, others observed no training effect (Table 1). Baldi *et al.* (30) reported that LV interventricular septal (IVS) and posterior wall thicknesses were 20-22% greater in older athletes than age-matched controls (65 years), whereas a 5% decrease in IVS was noted in the young trained compared with untrained. These data may suggest that athletes of the older population exhibit greater adaptations than younger individuals (26 years). Furthermore, prolonged dynamic exercises principally impose a volume overload challenge upon the LV and as a result, older endurance trained athletes have shown larger LV chamber diameters compared to their untrained counterparts (Table 1). Still, others have found similar end-diastolic diameters between trained and untrained groups (Table 1). An explanation for the contrasting findings is lacking at present, however, reductions in training stimulus (intensity, duration, volume) which occur with progressive ageing could contribute (58). Moreover, despite the suggestion

that trained-untrained differences in LV hypertrophy (LV mass) diminish or even disappear with advancing age in those beyond 45 years of age (59), the majority of studies have reported significantly increased LV mass (LVM) in trained individuals, expressed as absolute or allometrically scaled to indices of body size (Table 1). Still, whether differences between athletes and controls do progressively reduce with advancing age requires further study and clarification.

Continuous aerobic exercise training studies in previously sedentary, older males or females (53,60–66) have largely found unchanged LV morphology from pre-to-post training interventions ranging from 2-9 months. In contrast, three studies in older populations (all 68 years of age) found a statistically significant increase in LVM index of 5-18% following 4-12 months of dynamic exercise training (28,51,67) and suggested an eccentric remodelling (28,51). The lack of adaptations in the majority of studies are unlikely to be account for by an insufficient exercise intensity since specific high-intensity interval training (HIIT) programmes also observed unchanged morphology (53). In previously lifelong sedentary males (63 ± 5 years), Grace *et al.* (52) reported no changes in LV morphology following six weeks of supervised pre-conditioning exercise, which preceded a further 6 weeks of low-frequency HIIT. Furthermore, the training programme duration would likely elicit some influence on the magnitude of adaptation, however, since structural increases were observed after 4 months of football training (small sided games) in elderly men 65-75 years of age, (51) suggests adaptations can occur within short periods of high intensity intermittent training and thus, the programme duration may not be the sole determinant. Greater exercise stimulus including intensity, session duration and frequency, training programme duration, participant age upon recruitment or an interaction of these factors may be necessary to induce modifications within the LV structure. Additionally, and in consideration of the strong evidence from cross-sectional studies of greater LV mass in older trained adults with many

years of exercise training, it may be possible that adaptation occurs earlier in life and then maintained into older age with continued aerobic exercise training.

2.3 LV Systolic Function

LV systolic function is most commonly assessed using ejection fraction (EF) and is preserved at rest with healthy ageing (24,68). The majority of cross-sectional data report similar EF (Table 1) or fractional shortening (FS) (30,69–71) between older trained and untrained adults. With advancing age however, EF at maximal exercise is decreased while LV end-diastolic volume (LVEDV) increases which, together lead to unchanged SV index (24). Bouvier *et al.* (69) reported that EF was similar between master athletes and controls at rest, yet reported a significant training effect of greater EF at maximal exercise in the trained group. EF improved following 8-12 weeks interval training in older adults (36,53), which has not been observed following continuous exercise training (53,72). Indeed, the change in EF from pre-to-post exercise intervention was linearly related to the change in maximal oxygen uptake ($\dot{V}O_{2max}$) (53). Similarly, Fujimoto *et al* (28) reported 1 year vigorous exercise training improved $\dot{V}O_{2max}$ via favourable changes in maximal cardiac performance, without alterations in arterial-venous oxygen difference. However, another HIIT training study in older adults, of shorter duration and less frequency, reported no changes in EF (52), which may suggest that in addition to intensity, total exercise volume is important.

Alternate measures of LV systolic function include the mitral annular systolic tissue velocity (s') (73) which, may (30,35,36,38) or may not (31,37) demonstrate a decline concomitant with normal ageing. The majority of studies have found similar s' between trained and untrained adults (29,31,35,36,40,41,45). In addition to tissue velocities, newer methods of assessing LV systolic function have been developed, such as STE, with the advantage of being relatively angle independent and is not subjected to the tethering effect

(74,75). Global longitudinal strain (GLS) denotes shortening/deformation about its entire long-axis and when averaged across all LV wall segments is used as a measure of global systolic function (76,77). Compared with EF, GLS provides a greater means of directly assessing contractility and is a more sensitive marker of systolic (dys)function (77). Unlike EF, GLS decreases progressively from young to old in healthy participants (68,78). Although Schmidt *et al.* (40) found 12% greater (more negative) GLS in veteran football players (68 years) compared with age-matched controls, the general consensus from other observational studies is a lack of training effect on GLS in ageing athletes (29,32,45,79). The participants recruited could explain why Schmidt *et al.* (40) observed an effect while others did not. The veteran footballers (68 years) were still regularly competing throughout the year (26 ± 12 soccer matches) and compared with participants in the other studies, were the oldest age and had the longest training history (52 ± 11 years). Exercise stimulus may also be important, a training study from the same group found that following 12 months of football specific training in previously lifelong sedentary senior (68 years) males, EF increased and GLS increased (more negative) by 8% (51). Moreover, recently Howden *et al.* (80) reported a lifelong (at least 25 years) exercise training dose of at least 4 sessions per week in seniors (>60 years) prevented the age-related decline in GLS. Following adjustment for LVEDV however, the training effect was abolished which, as noted by the authors, highlights the importance of training related changes in LV filling volume in preserving systolic function with ageing (80). Of particular note, trained seniors showed the largest decrease in GLS following preload reduction and at similar EDV, GLS was significantly lower in the trained than untrained adults (80). Taken together, exercise training may improve systolic function, yet more longitudinal studies are required with the inclusion of GLS which, is sensitive to ageing and potentially exercise training, alongside conventional measures of structure and systolic function.

Beyond longitudinal shortening during contraction, the LV also rotates along its long-axis (76). Systolic twist determined by the opposing rotations at the base and apex in clockwise and counter clockwise directions, respectively (76), increases stepwise with age in a general population of healthy individuals (55). Maufrais *et al*. (35) documented lower resting twist in senior athletes compared with controls, while two studies observed no training effect (34,54). With the transition from rest to exercise, Lee *et al.* (34) found middle-aged aerobic athletes were able to increase twist compared with controls, whereas Maufrais *et al.* (54) observed no differences between training levels during submaximal cycling. In younger individuals LV twist increases with submaximal exercise (81,82), which is closely coupled with exercise SV (83). The ability to increase LV twist in older athletes in response to a physiological exercise stress would suggest a greater functional capacity to accommodate the heightened cardiovascular demands by modulating LV output. Nonetheless, further assessment of twisting mechanics in older athletes both at rest and during exercise could provide additional, insightful information to advance our understanding of systolic functioning following long term exercise training in maters athletes.

3 Conclusion

While the preponderance of studies examining cardiac and vascular responses in clinical populations in understandable it is likely that morbidity imposes additional reductions in cardiovascular function. Nevertheless, there is growing evidence that undertaking regular exercise training results in improved indices of diastolic performance, and that this superiority in myocardial relaxation is maintained into the eighth decade of life. In addition, lifelong exercisers exhibit moderate remodelling to support greater SV and cardiac output. However, differences in systolic function are less clear, however, much of this comparative

data has been acquired at rest, and it is possible that larger differences may be evident during exercise, as the greater functional reserve of masters athletes becomes apparent. There is a need for more observational studies, to include exercise measures, as well as wider use of novel imaging technologies within this cohort.

The contrast of sedentary controls and masters athletes provides a useful model to investigate cardiovascular function during advancing age. The superior findings in those who have sustained exercise training into old age, suggest that the declines assumed to occur with age are less precipitous than previously suspected, and that while some functional impairment seems inevitable, partaking in regular exercise results in a significant slowing in the rate of decline. However, there is a need for more research to help elucidate the mechanisms of true age-related decline, and the mechanisms of decline due to sedentariness. In addition, more data are needed to determine the most effective prescription to improve cardiac function.

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Table 1 Summary of studies including echocardiographic derived left ventricular structure, systolic and diastolic function in athletes and controls

Study (year of publication)	Participant characteristics		Echocardiographic measures						
	Sport (Gender- M/F)	Age (years)	Wall thickness			LVEDD	LVM	E/A	EF
			IVS	PWT	MWT				
Baldi et al. (28)	Controls (M) Aerobic (M)	65.7 ± 3.7 65.2 ± 4.2	↔	↔		↑	↑ ↑ *	↔	
Bhella et al. (27)	Controls (M+F) Endurance (M+F)	68.8 ± 5.1 67.8 ± 2.9						↔	
Bohm et al. (43)	Controls (M) Runners, rowers, triathletes (M)	46.0 ± 9.0 47.0 ± 8.0	↑	↑		↑			
Bouvier et al. (57)	Controls (-) Orienteers, runners (M)	74.9 ± 2.4 72.8 ± 2.9	↔	↑		↔	↔ *	↑	
Carrick-Ranson et al. (29)	Controls Cyclists, runners, dual/triathletes (M)	66.0 ± 5.0 66.0 ± 4.0				↔	↑ ↑ *	↑	
Child et al. (69)	Controls (M) Runners (M)	56.3 ± 7.8 53.7 ± 10.6	↑ *	↑ *		↑ *	↑ *		
Cottini et al. (37)	Controls (-) Aerobic (-)	61.0 ± 7.0 60.0 ± 10.0	↔	↔		↔		↑	↑
D'Andrea et al. (19)	Controls (M) Swimmers (M)	47.4 ± 2.2 48.2 ± 3.4	↔	↔		↑	↑ *	↑	
Di Bello et al. (70)	Controls (M) Runners (M)	69.7 ± 8.4 65.7 ± 7.1	↑	↑		↔ ↔ *	↑ ↑ *	↑	↔

Study (year of publication)	Participant characteristics		Echocardiographic measures						
	Sport (Gender- M/F)	Age (years)	Wall thickness			LVEDD	LVM	E/A	EF
			IVS	PWT	MWT				
Donal et al. (30)	Controls (M) Cyclists (M)	58.9 ± 8.6 61.5 ± 5.6	↑	↑		↔	↑ *	↔	↔
Douglas and O'Toole. (41)	Controls (M+F) Ultra-endurance (M+F)	65.0 ± 6.0 58.0 ± 6.0		↔ ↔		↑ ↑ *	↔ ↔ *	↑	
Fleg et al. (40)	Controls (M) Runners (M)	63.0 ± 6.0 65.0 ± 8.0	↔ *	↔ *		↔ *	↔ *	↔	
Galetta et al. (85)	Controls (M) Runners (M)	66.9 ± 4.6 67.6 ± 4.5	↑	↑		↑	↑ ↑ *	↔	↔
Galetta et al. (39)	Controls (M) Runners (M)	68.3 ± 3.2 69.4 ± 3.8	↑	↑		↑	↑ ↑ *	↑	↔
Gates et al. (31)	Controls (M) Aerobic (M)	65.0 ± 6.6 68.0 ± 6.9			↑ *	↑ *	↑ *	↑	
Giada et al. (86)	Controls (M) Cyclists (M)	58.0 ± 6.0 55.0 ± 5.0	↑ *	↑ *		↑ *	↑ *	↔	↔
Grace et al. (49)	Controls (M) Endurance (M)	62.7 ± 5.2 61.1 ± 5.4	↔	↔		↔	↔ ↔ *	↔	↔
Jungblut et al. (87)	Controls (M) Runners (M)	69.0 ± 3.0 69.0 ± 5.0	↔	↔		↑	↑ *	↔	
Kozakova et al. (88)	Controls (M) Marathoners, triathletes (M)	46.5 ± 16.0 53.1 ± 20.0	↑	↑		↔	↑ ↑ *		
Lee et al. (32)	Controls (M) Cyclists, triathletes, speed-skaters (M)	54.8 ± 4.3 53.8 ± 4.1	↔	↔		↔	↑ *	↔	↔

Study (year of publication)	Participant characteristics		Echocardiographic measures						
	Sport (Gender- M/F)	Age (years)	Wall thickness			LVEDD	LVM	E/A	EF
			IVS	PWT	MWT				
Lindsey and Dunn (89)	Controls (M) Runners (M)	52.0 ± - 52.0 ± 11.4	↑	↑		↑	↑ *	↔	
Maessen et al. (79)	Controls (M) Endurance (M)	58.0 ± 7.0 61.0 ± 7.0						↔	
Matelot et al. (28)	Controls (M) Runners, cyclists (M)	59.0 ± 3.0 62.0 ± 3.0	↔	↑		↔ ↑ *		↔	↔
Maufrais et al. (33)	Controls (-) Runners, triathletes, cyclists (M)	56.0 ± 6.0 54.0 ± 7.0			↑	↑	↑ *	↑	
Maufrais et al. (51)	Controls (M) Cyclists (M)	55.0 ± 8.0 57.0 ± 8.0				↑	↑ *		
Miki et al. (90)	Controls (-) Cyclists (-)	49.0 ± 7.6 49.4 ± 6.4			↑ *	↑ *	↑ *		
Molmen et al. (34)	Controls (M) Cross-country skiers (M)	71.7 ± 1.3 74.3 ± 1.8						↔	↔
Nishimura et al. (91)	Controls (M) Bicyclists (M)	46.9 ± 3.3 45.6 ± 2.3	↑	↑		↑	↑		↓
Northcote et al. (92)	Controls (M) Runners (M)	56.0 ± 7.0 56.0 ± 7.0	↔ ↑ *	↑ ↑ *		↔ ↔ *	↑ ↑ *		
Nottin et al. (35)	Controls (M) Cyclists (M)	55.9 ± 4.1 58.6 ± 4.8	↔ *	↔ *		↑ *	↔ *	↑	↔

Study (year of publication)	Participant characteristics		Echocardiographic measures						
	Sport (Gender- M/F)	Age (years)	Wall thickness			LVEDD	LVM	E/A	EF
			IVS	PWT	MWT				
Olsen et al. (36)	Controls (M) Runners (M)	66.3 ± 3.8 65.0 ± 4.6			↑ ↑ *	↑ ↑ *	↑ ↑ *	↑	↔
Prasad et al. (22)	Controls (M+F) Marathoners, triathletes, middle-distance runners (M+F)	69.8 ± 3.0 67.8 ± 3.0			↔		↑ *	↑	↔
Seals et al. (93)	Controls (M) Runners (M)	63.0 ± 3.0 64.0 ± 6.0		↑ *		↑ *			
Schmidt et al. (38)	Controls (M) Soccer players (M)	68.2 ± 3.2 68.1 ± 2.1	↔	↔		↔	↔	↔	↑
Takemoto et al. (42)	Controls (M+F) Runners (M+F)	60.0 ± 5.0 60.0 ± 7.0						↑	
Vianello et al. (94)	Controls (M+F) Marathoners (M+F)	57.0 ± 10.0 58.0 ± 6.5	↑	↑		↑	↑ *	↑	↓

M, male; F, female; IVS, interventricular septal thickness; PWT, posterior wall thickness; MWT, mean wall thickness; LVEDD, left ventricular end-diastolic diameter, LVM, left ventricular mass; E/A, early-to-late mitral inflow velocity; EF, ejection fraction. *, indicates allometrically scaled indices; ↑, significantly greater in athletes as reported by study; ↓, significantly lower in athletes as reported by study; ↔, no significant difference between athletes and controls as reported by study. Data presented as means ± standard deviation.